and informed approach to long term catheterisation and are eager to support further research into specific aspects of catheter management. Two short booklets are available from Bard Limited for patients and those responsible for their care, and further information will be gladly given to any doctor heeding Mr Kinder's call.

1 Blannin JP, Hobden J. The catheter of choice. *Nursing Times* 1980;78:438-40.
2 Kennedy AP, Brocklehurst JC. The nursing management of patients with long-term indwelling catheters. *Journal of Advanced Nursing* 1982;7:411-7.
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Alcohol and violence

Mr M CAREW-MCCOLL, Mr T F BEATTIE, and Dr J W GERRARD (Accident and Emergency Department, Royal Preston Hospital, Preston PR2 4HT) write: We share the concern expressed by Mr D W Yates and Ms H Chambers about the number of assaults that they see in their accident department and the strong relation between such assaults and alcohol (4 April, p 901). We recently reviewed the problem in this hospital. Assaults constituted 2·5% of our workload, alcohol was a factor in 80% of assaults, and we estimated that the cost to our health authority in 1986 was £100 000.

Drug points

Lead poisoning in heroin addicts

Dr S MONTEFORT and colleagues (St Luke's Hospital, Gwardamangia, Malta) write: In Malta over the past year we have encountered several cases of lead poisoning in heroin addicts. A 21 year old man, a known heroin abuser, presented in December 1985 with abdominal pain, distension, and constipation. Abdominal radiography showed severe colonic distension, and opiate induced colonic pseudo-obstruction was diagnosed and colonoscopic decompression performed. He was, however, found to have severe sideroblastic anaemia and a blood lead concentration of 1010 μg/l (on absorption spectrophotometry the 90th centile for normal subjects is 343 μg/l).[1] There was no history of occupational exposure to lead, and a sample of heroin provided by the patient contained 21 mg lead/g powder. The sample also contained calcium and silicon (thought to be due to talc) and small traces of several other metals, including copper, zinc, iron, and chromium. Since screening was subsequently introduced 46 episodes of lead toxicity have been diagnosed in 22 known heroin abusers (with up to four admissions each). There were 19 men and three women, age range 20-33. All patients had gastrointestinal symptoms on presentation, including abdominal pain (10%), constipation (90%), nausea or vomiting (95%), and abdominal distension (30%). Weakness and pallor were also frequent (90%); one patient suffered from tremulousness. All patients had anaemia, with haemoglobin concentrations ranging from 65 g/l to 120 g/l. Lead concentrations ranged from 506 μg/l to 3648 μg/l. Anaemia was treated with blood transfusion, and intravenous edetic acid, with or without oral penicillamine, was used to lower blood lead concentrations. The patients became asymptomatic, with oral methadone, and in some cases clonidine, being given for withdrawal symptoms. Despite the knowledge that the heroin was contaminated 30% of the patients were readmitted within weeks with a recurrence of lead toxicity and admitted to further heroin abuse, many having also stopped taking penicillamine.

In a known intravenous drug abuser the combination of anaemia and gastrointestinal symptoms should suggest the association of lead toxicity and intravenous heroin abuse. Lead poisoning has been reported in addicts who injected themselves with ground up lead and opium pills.[2] In our patients, however, lead salts, probably acetate or nitrate, were added to heroin as a white powder to increase the selling weight. Talc may also be used to dilute heroin, and poisoning with non-infectious contaminants is becoming increasingly recognised as a complication of intravenous drug abuse.[3,4]

Treatment is difficult, because poor compliance hinders attempts to reduce the total body lead stores with oral chelating agents. In addition, awareness of the risks of lead poisoning did not prevent many of these drug abusers from reusing a known contaminated source.

1 Friberg L. *Assessment of human exposure to lead: comparison between Belgium, Malta, Mexico and Sweden.* Stockholm: Karolinska Institute, 1985.
2 Beattie AD, Briggs JD, Canavan JS, Doyle D, Mullin PJ, Watson AA. Acute lead poisoning: five cases resulting from self-injection of lead and opium. *Q J Med* 1975;44:275-84.
3 Langston JW, Ballard P, Tetrud JW, Irwin I. Chronic parkinsonism in humans due to a product of meperidine-analog synthesis. *Science* 1983;219:970-80.
4 Brauner D, Sherer R. Talcosis in AIDS victims? *Am J Clin Pathol* 1984;81:145.

Prescribing in pregnancy: rifampicin

Dr NALIN K ASHUBODHA (Mahabuthgamuwa, Sri Lanka) writes: Dr Richard Wise's article on prescribing antibiotics in pregnancy (3 January, p 42) is particularly pertinent to the tropical world, where antibiotics are often misused, overused, or underused. Rifampicin was used in 11 pregnant patients with leprosy (age range 19-31) in the pilot multidrug therapy trials at Shashamene, south Ethiopia.[1] Nine of the 11 were paucibacillary patients, who had been receiving dapsone 100 mg orally daily and rifampicin 600 mg orally monthly, with supervision, for six months. The remaining two patients, who were multibacillary, had been receiving dapsone 100 mg daily, clofazimine (Lamprene) 100 mg daily, rifampicin 600 mg monthly, and clofazimine 300 mg monthly for two years.[2] Most of these patients were included in the study without our knowing that they were pregnant. All of the patients were given strict contraceptive advice during the test period, and a special family planning clinic was set up for them. When we began to suspect that they were pregnant the first trimester was already over. At the end of the first trimester I suggested that the patients should continue to take rifampicin 600 mg monthly with the other antileprotic drugs for the respective test periods. A good history, particularly of gastrointestinal symptoms, fevers, and shortness of breath, and a general physical examination with skin examination in good daylight were mandatory. Laboratory investigations included haemoglobin tests, differential and total white blood cell counts, urine analysis, and liver function tests.[3]

No toxic effects are encountered in non-pregnant patients with leprosy who receive doses of rifampicin monthly. Similarly, we found no toxic effects in pregnant patients with leprosy who received 600 mg rifampicin monthly.

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2 World Health Organisation. Chemotherapy of leprosy for control programmes. *WHO Tech Rep Ser* 1982; No 675.
3 World Health Organisation. A guide to leprosy control. Geneva: World Health Organisation, 1980.

Ventricular tachycardia after self poisoning with dichloralphenazone and diazepam

Dr J A LOCKTON (Wythenshawe, Manchester) writes: There have been several reports of ventricular arrhythmia after self poisoning with chloral hydrate[1-3] and supraventricular tachycardias in children receiving chloral hydrate after cardiac surgery.[4] Arrhythmias have not been reported with the related drug dichloralphenazone.

A 30 year old woman was prescribed 90 tablets of diazepam 10 mg and 90 tablets of dichloralphenazone 650 mg by her general practitioner. She was admitted to hospital 10 days later after taking an overdose of the tablets. Cardiac monitoring showed multiple ectopic beats and short, self terminating runs of ventricular tachycardia. No treatment was required and she recovered uneventfully. Urine toxicology on a sample taken 12 hours after admission showed dichloralphenazone and its metabolites. She was readmitted three months later, again deeply unconscious. She had been found with an empty packet of dichloralphenazone tablets, having collected a further prescription for diazepam and dichloralphenazone tablets two days earlier. Ventricular tachycardia recurred, this time with cardiovascular collapse. This failed to respond to defibrillation after boluses of lignocaine 100 mg and bretylium but reverted to sinus rhythm during a half hour infusion of amiodarone 375 mg. Subsequently she recovered. Clinical examination and electrocardiography during each admission showed no evidence of underlying cardiac disease.

Ventricular tachycardia in our patient was probably due to dichloralphenazone as this drug is metabolised to chloral hydrate, with which ventricular tachycardia has been described. The Leeds poison centre confirmed that there had been reports of ventricular tachycardia with dichloralpenazone, although none have been published. The mechanism is thought to be sensitisation of the myocardium to endogenous catecholamines and the treatment of choice β blockade. There have been no such reports concerning diazepam even though it is commonly taken in cases of self poisoning.

I thank Dr N Brookes (consultant cardiologist) for his help in preparing this report and Dr J J Manns (consultant general physician) for permitting me to report this case.

1 Marshall AJ. Cardiac arrhythmias caused by chloral hydrate. *Br Med J* 1977;ii:994.
2 Wiseman HM, Hampel G. Cardiac arrhythmias due to chloral hydrate poisoning. *Br Med J* 1978;ii:960.
3 Brown AM, Cade JF. Cardiac arrhythmias after chloral hydrate overdose. *Med J Aust* 1980;i:28-9.
4 Hirsch IA, Zaunder HL. Chloral hydrate: a potential cause of arrhythmias. *Anaesth Analg* 1986;65:691-2.

Pinpoint pupils in mianserin overdose

Dr G N FULLER and Ms JANET CLARBOUR (St Stephen's Hospital, London SW10 9TH) write: Pinpoint pupils in a drowsy patient who has taken an overdose are usually associated with opiates.[1] We describe a case in which these findings were due to mianserin. A 33 year old woman was seen two hours after taking 25-40 tablets of mianserin 30 mg and half a bottle of rum. She denied taking any other drugs and was receiving no regular medication. On examination she was drowsy, but able to answer questions, and had pinpoint pupils. An electrocardiogram confirmed her tachycardia of 120 beats/min and showed no conduction defect. Naloxone 2·4 mg was administered intravenously with no response in either pupil size or level of consciousness. Gastric washout was performed. Six hours after the overdose she was alert and her pupils were 2 mm in diameter. Twenty four hours later they were 4 mm in diameter and reacting normally to light and accommodation. Toxological analysis subsequently confirmed that no other agents had been taken. Two hours after overdose mianserin concentrations were 0·39 mg/l, desmethylmianserin 0·07 mg/l, and ethanol 3·00 mg/l. Therapeutic concentrations of mianserin (plus desmethylmianserin) are 0·1 mg/l, and serious toxicity is associated with concentrations above 0·5 mg/l.

Pinpoint pupils have been reported in mianserin overdose before, once in mianserin alone and twice in combination with other drugs.[2] In studies of normal volunteers[3] and depressed patients[4,5] mianserin produced a statistically significant reduction in pupil diameter of 1·0-1·5 mm. The mechanism of this effect is not clear, but at therapeutic levels it was suggested that it was either centrally mediated or from interactions with receptors other than cholinergic or adrenergic.[5] Mianserin in overdose is a possible cause of pinpoint pupils unresponsive to naloxone.

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3 Kopera H. Anticholinergic and blood pressure effects of mianserin and placebo. *Br J Clin Pharmacol* 1978;5:29-34s.
4 Wilson WH, Petrie WM, Ban TA. Possible lack of anticholinergic effects with mianserin: a pilot study. *J Clin Psychiatry* 1980;41:63-5.
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